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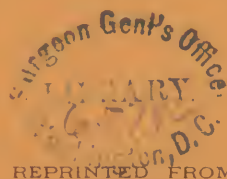
Peculiar Paraplegiform Affection.

(TETANOID PSEUDO PARAPLEGIA.)

BY

E. C. SEGUIN, M.D.

LECTURER ON DISEASES OF THE NERVOUS SYSTEM AT THE COLLEGE OF PHYSICIANS AND SURGEONS, NEW YORK.



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DESCRIPTION OF A PECULIAR PARAPLEGIFORM AFFECTION.

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THE condition to which I desire to call attention is one which cannot be of very rare occurrence, as, in course of three years, I have met with five examples of it. No doubt many readers will at once remember having observed precisely similar symptoms.

This form of false paraplegia (using this word as implying the existence of paresis or akinesis in the lower limbs) is characterized by impairment of the functions of the lower extremities, when the patient is in the erect posture, without any loss of power in these parts. Further analysis shows that the seeming paraplegia is dependent upon tonic spasm of the muscles of the lower limbs. As negative characters we have absence of the symptom ataxia, and often, also, preservation of sensibility.

The clinical aspects of a case of tetanoid pseudo-paraplegia are the following: The patient complains of having nearly lost the use of his lower limbs (he may speak of great "loss of power"); of having various abnormal sensations in them, and of experiencing trouble in the evacuation of his feces and urine. When the patient is told to get up and walk, he rises with difficulty from the bed or chair, assisting himself with his hands. On getting into a perpendicular position, with or without the aid of a stick, he oscillates a good deal, and seeks to re-establish his equilibrium by separating his feet, and bending his body forward. In this posture the knees remain extended, and the feet are not everted as in health; they are often, on the contrary, turned inward. The attempted steps are peculiar. The feet are not dragged along as in ordinary cases of incomplete paraplegia, nor are the knees much flexed, and the feet brought down violently as in locomotor ataxia. There is none of the outward projection of the entire limb, so characteristic of the latter disease. The limbs remain extended, and the feet are simply pushed along the floor; the slight raising of the soles from the support being accomplished by a movement involving the entire lower extremity. The tendency of the great toe is downward and inward, thus producing, or tending to produce, a partial crossing of the limbs, and tripping. If during the efforts the observer feel the patient's muscles, he will find them firmly contracted.

If the patient be seated, or made to lie upon a bed, and the strength of his lower limbs tested in the usual way, *i. e.*, by bidding him resist attempts at passive flexion or extension of certain articulations, the muscular power will be found almost up to the normal limit, if not quite so, in every part of the lower half of the body. There is not, necessarily, any weakness of the abdominal muscles. There is, consequently, no paresis present, and we cannot correctly speak of the case as one of paralysis. The reflex power of the lower limbs, tested in these two positions, is found to be much exaggerated, and a state approaching spinal epilepsy (a mixture of clonic and tonic spasms) may be developed by the examination. Caloric appears to excite these reflex actions most readily.

The state of the bladder and rectum is sometimes peculiar (Cases III. and IV.).

The urine does not dribble away, and exhibits no pathological alkalinity. If passed involuntarily, it is at intervals, and by jets; a normal desire to urinate occurring. Usually the patient is simply obliged to hurry the evacuation of the viscus, or an emission of urine by reflex action will take place very quickly after the sensation of fullness has been perceived. One of the patients (Case IV.) expressed

the state of affairs very well by saying, "Now that I'm better, I can go the length of the ward before letting go, but no further." The bowels are, as a rule, constipated, but when feces descend into the rectum, a rapid emptying of the organ is inevitable. It appears to me reasonable to suppose that this morbid excitability of the rectum and bladder existed in all the cases at some period or other. Retention of urine—a truly parietic trouble—is apt to develop as the case progresses.

As above stated, sensibility is not necessarily impaired, and in only one case (Case I.) was there much anæsthesia. In two cases (Cases II. and III.) sensibility remained normal. It is also noteworthy that the existence of considerable cutaneous anæsthesia (Case I.) did not at all interfere with the production of reflex movements.

In no case could true ataxia be made out. The muscular sense appeared normal, and what inco-ordination existed was due to spasmodic action of the adductor muscles. It will be remembered that in ataxia (the symptom) affecting the lower extremities, exaggerated action of the abductor muscles is present.

The pathological condition to which these symptoms seemed, in all the cases, allied, appeared to be compression of the anterior part of the spinal cord in the dorsal or cervical region. In three instances (Cases I., II., and V.) this can hardly be questioned, since cyphosis existed. In Cases III. and IV., I admit that the diagnosis of tumor is not established in the most conclusive way, but as these patients are still under my observation, I may at some future time be able to clear up the uncertainty. As things now stand, I believe that I am authorized to conclude that the peculiar false paraplegia caused by reflex movements, and to which I venture to give the name of tetanoid pseudo-paraplegia, is to be looked upon as a symptom of moderate compression of the spinal cord at some point above the lumbar enlargement. I do not wish to be understood as denying that tetanoid pseudo-paraplegia may occur in cases of functional disturbance of the spinal axis. Although I have seen no such case recorded in the publications accessible to me, I have no doubt that a similar or analogous state may be observed in hysteria. It appears probable to me, however, that in cases of increased spinal excitability, without lesion, the spasms would be more clonic (saltatory) than tonic (tetanoid) in character.

The pathogeny of this symptom is similar to that of the closely allied (often coexistent) group of symptoms called spinal epilepsy. In the first place, as a cause of increased reflex action of the spinal cord must be reckoned the diminution of the cerebral influence

brought about by compression of the cord. This is in accordance with what experimentation upon the lower animals teaches us. It is a pretty generally admitted view, that the cerebral influence moderates the motricity of the spinal cord. Another element in the production of these reflex spasms I believe (with Dr. Brown-Séquard,* when he speaks of spinal epilepsy) to be congestion of the spinal cord below the lesion.

This symptomatic group evidently belongs to the class of hyper-æneses (Romberg), and therefore it may be interesting to determine its relationship with some other varieties of increased muscular action.

Its most closely allied congener is spinal epilepsy. This appellation was given by Dr. Brown-Séquard to a combination of tonic and clonic spasms affecting paralyzed parts in certain affections of the spinal cord. He describes it in these terms: "Whether spontaneously, or after an external irritation (such as a shock, or a pressure on some muscles, tickling the sole of the foot, or the passing of a catheter into the urethra), the lower limbs are often moved violently or become perfectly stiff; sometimes they are drawn up forcibly in a state of flexion, the back part of the foot pressing against the hip-joint; sometimes the thighs are drawn violently one against the other by a spasm of the adductor muscles, and they press very hard against the testicles; in other cases the flexor and extensor muscles contract alternately with great violence, and, after a few minutes of great shaking, a rigid condition appears, which, after a time, is followed by relaxation and quietness."† Dr. Brown-Séquard believes that "this spasmodic affection of the paralyzed legs is the result of the morbid increase in the vital properties of the dorso-lumbar enlargement of the spinal cord, owing to two causes: 1st, the congestion of that part of the cord; 2d, the accumulation of power in that part of the cord, in consequence of its not being any more under the action of the will." He states that the pathological conditions which, according to their localities, may produce this symptom in man, are localized myelitis, tumors pressing upon the cord, fracture and dislocation of the vertebrae; and that by section of the spinal cord he can easily produce this condition in animals. The same group of symptoms, a combination of tonic and clonic spasms occurring under similar circumstances, had many years before attracted the attention of several observers, and Dr. William Budd has left on record an admirable description of these spasms as observed by him prior to 1839.‡ Jaccoud§ consid-

* Lectures on the Diagnosis and Treatment of Paralysis of the Lower Extremities. Phila. 1861 p. 60.

† Brown-Séquard, op. cit., p. 59.

‡ Médico-Chirurgical Transactions. Vol. xxii., p. 153. 1839.

§ Des paraplegies et de l'ataxie du mouvement. Paris, 1864, pp. 484, 488.

ers exaggerated reflex power in paralyzed limbs in general, and the form of hyperkinesis now under consideration (spinal epilepsy) in particular, as a positive sign of organic paraplegia.

A noteworthy variety of spinal epilepsy consists in a succession of clonic spasms (trembling) of limited range affecting the paretic or akinetic limbs. In cases of partial, insulated cerebro-spinal sclerosis (and other morbid conditions of the spinal cord), this trembling may be excited by irritation of peripheral nerves; and forcibly flexing the foot (patient being in recumbent position) seems to have special efficacy. A similar movement may be produced in healthy individuals by insufficient and ill-placed support of an extremity: a foot, for example.

The spasm which constitutes the most important element in the obscure affection known as writer's cramp is analogous to tetanoid pseudo-paraplegia. Here the patient, while writing the first few words, experiences no marked difficulty, but after the spinal cord has been acting for some time, a spasm, more or less tonic in character, affects the flexor muscles moving the thumb and fingers, and there ensues an illegibility in the writing, or an utter impossibility to hold the pen. The same spasm of the flexors occurs when the patient is using his fingers for other purposes, such as holding a cup or saucer; never spontaneously. We thus have hyperkinesis determined by the action of a peripheral irritation upon a functionally diseased nervous centre.

There is, it seems to me unquestionable, a spasmodic element in the complex group of symptoms which constitutes the disease known as locomotor ataxia. In the first place, as believed by Brown-Séquard,* the conservation of force in the lower limbs, in the paraplegic forms of this disease, is only apparent. I am ready to admit with him, that a certain degree of paresis exists in the affected parts, but that the methods employed for testing the degree of volitional force really develops a degree of reflex (morbid) power which, after the first moments of the examination, conceals whatever loss of power may previously have existed, and causes even an abnormal degree of muscular strength. Secondly, in the symptom ataxia, I have for some time believed that the characteristic disharmony in the action of various muscular groups in the typical stages of the disease locomotor ataxia, was due not so much to diminished nervous influence in the muscles overcome by their antagonists, but in increased motricity sent to the over-acting muscles. Thus, in the jerking, externally projected steps there is an overbearing action of the abductors and extensors, and this is due, in my opinion, to the reception of abnormal increased motricity by these muscles; a motricity developed in a reflex way by the exercise of the limbs (see above, writer's cramp).

* Oral Communication, 1869.

I am aware that recent writers are inclined to consider the disharmonious action above referred to as wholly dependent upon varying degrees of impairment of the muscular sense. If this be so, why should the disturbance affect definite muscular groups?

Certain forms of contracture of the paralyzed limbs in hemiplegia (of cerebral origin) bear a very close resemblance to the false paraplegia I have endeavored to describe. There is now a male patient under my care at the Epileptic and Paralytic Hospital, Blackwell's Island, who experienced months ago an ordinary apoplectic stroke, followed by right-sided hemiplegia (including face), and temporary aphasia. He has recovered some degree of voluntary motion in the palsied limbs, but suffers much from contracture of the arm and hand. The resident physician, Dr. Bruce, the nurses, and the patient himself assure me that at night and in the early morning, before exposing the parts to the air, or attempting to rise, the hand lies quite open and relaxed, and that no stiffness whatever exists at the elbow-joint. The contact of air, however, and, more surely, the acts of rising and stepping upon the cold floor provoke a spasm which in a few minutes reaches its maximum. When this is at its highest degree of tension (as it is during my visits) the forearm lies across the chest (patient sitting in a chair or walking), the elbow being bent about at right angles; the wrist is somewhat flexed, and the finger-nails are forced into the palms of the hand. The observer's efforts to overcome this contracture only increase it, and the same is true of the patient's own volitional efforts, and of his using the other limbs. The right lower extremity is moderately stiffened, in extension, during waking hours. The flexors and adductors of the upper extremity have not suffered in nutrition, while the overpowered (stretched) extensors are in a state of unmistakable atrophy, and have lost electro-muscular contractility. The pathological physiology in this case I believe to be precisely similar to that explained when speaking of tetanoid pseudo-paraplegia. The principal cause of spasm (increased reflex power) in both cases is the separation of the spinal axis from the cerebrum: in the false paraplegia the cutting off occurs somewhere in the spinal cord, while in hemiplegia it happens at the junction of the upper end of the spinal axis with the cerebrum (corpus striatum).

I wish to add a few words concerning a form of "stiffness" of the lower limbs which is much complained of by patients having congestion (?) of the spinal cord and its meninges. This is a purely subjective sensation which accompanies the numbness and formication which form such prominent features in these cases. The "stiffness" as well as the numbness are worse when the subject is lying down or sitting, and are greatly felt during the first efforts at movement. Contrary to

what obtains in spasmodic false paraplegia, this feeling grows less marked after the patient has taken active or passive exercise. There is no real (objective) rigidity, and reflex movements are not necessarily modified from the healthy standard.

As regards bibliography I can say but little. Only one writer, to my knowledge, seems to have noticed and described a condition similar to the one forming the subject of this contribution. I refer to Jaccoud,* who, in his valuable work on various forms of paraplegia gives a page and a half to what he calls false paraplegia due to exaggerated spinal excitability. He does not refer to any cases, nor does he give any account of the pathological conditions accompanying the symptom. He undoubtedly has seen cases similar to mine. I have come across a reference which may be thought to indicate that the physician referred to had seen and described spasmodic false paraplegia. E. Goupil† made (ever published?) a classification of hysterical paraplegias, embracing the following varieties: 1st. Hysterical paraplegia, due simply to muscular weakness; 2d. Hysterical paraplegia, produced by the extreme pain caused by reflex action and movement; 3d. Hysterical paraplegia produced by loss of muscular sensibility. While admitting that Goupil's second variety bears a certain resemblance to tetanoid pseudo-paraplegia, I would recall that in the cases I am about to detail, pain on movement was not a feature, and that hysteria had nothing to do with any one of my five instances. The older and more recent treatises and monographs upon diseases of the nervous system, with the above exception, contain no reference to the condition I have described.‡

CASE I.—By the kindness of Dr. Gustavus A. Sabine, I had the opportunity of studying the symptoms presented by Mr. P., an Englishman, aged 46 years. About six weeks before the consultation (April 6, 1872), patient noticed while at first walking in the morning a slight degree of numbness and formication with "loss of power" in the lower extremities. The numbness affected all the parts below the knees, and to a much less degree the anterior surface of thighs. At same time, or shortly after, he observed twitching of the lower limbs at night. These three symptoms, formication, "loss of power," and twitching, progressively increased, until he now walks with difficulty, even when aided by a stick. Yesterday was obliged to ask assistance to cross a busy thoroughfare. The bowels have been costive and the urine hard to pass. Since three weeks has felt as if a band were tightly drawn around the lower part of the abdomen. Lower limbs have moderately wasted. The "loss of power" has rapidly increased in the last three days.

* Jaccoud, op. cit. pp. 469-471.

† Cited by Leroy: *Des paralysies des membres inférieures*. Paris, 1856; p. 210.

‡ After the above had been printed, I had the opportunity of reading Hallopeau's interesting thesis, entitled *Des accidents convulsifs dans les maladies de la moelle épinière*, Paris, 1871, in which I find a case of false paraplegia caused by spasm, well described (Case III., p. 50). The author correctly appreciates the significance of the symptom, but has not called attention to it specially in his remarks.

The examination shows nothing abnormal about the upper part of body excepting the fact that the pupils are extremely minute.* The co-ordination of the upper extremities is perfect. The patient walks in a peculiar way. He leans firmly upon a stick, and takes very short steps, with limbs almost perfectly rigid. There is neither distinct jerking outward of the feet, nor dragging; but patient staggers much. Stands with feet somewhat separated, and without stick oscillates a good deal. The walk is not made different or worse by closure of eyes. Strength of lower limbs, tested in sitting and recumbent postures, shows very slight, if any, impairment. Movements well co-ordinated. Some disturbance of sensibility; superficial contact not normally perceived, impressions of pain retarded and metamorphosed into burning. Localizes impressions well; and sense of temperature is normal. Reflex movements exaggerated. Lower abdominal muscles weak; cause of constipation and slow micturition. No spontaneous pain in back or limbs. Deep pressure reveals obscure tenderness on level of fifth and sixth dorsal vertebræ. Patient states that stiffness in limbs and back is worst in early morning, and is somewhat relieved by exertion. Twitching of legs increased; often has alternations of clonic and tonic spasms (spinal epilepsy). On the 23d, a second examination shows continuance of numbness and stiffness. Cramps decidedly less. Rather more impairment of sensibility; pinching produces severe burning. Patient loses his limbs in bed. Once attempted to rise in dark, and failed to "strike bottom" with his feet; found himself on his knees. Is unconscious (eyes closed) of passive movements below hips. Co-ordinates well. May 3d. The obscurity of the case is to-day cleared up by the discovery of a slight but distinct angular curvature of the spine, caused by projection of spinous processes of fifth and sixth dorsal vertebræ. Deep pressure produces pain. A few days later the patient started for England.

CASE II.—J. H., male, aged 32 years, in my service at the Epileptic and Paralytic Hospital, Blackwell's Island. Patient was first admitted in March, 1869, discharged, and readmitted in July, 1870. Two histories of the case are on record, which differ somewhat as to the mode of reception of the injuries which produced various symptoms; there is no conflict, however, on the points which render the case interesting in the present connection, viz., on the state of the spinal column and of the lower limbs. I will abridge from both accounts, one of which was written at the bed-side under my own dictation.

Eighteen years ago a chimney fell upon patient and produced a compound fracture of the skull, necessitating the removal of large pieces of bone. No paralysis resulted from wound or operation. Now bears a large scar on left side of vertex, six inches in length (antero-posterior), $\frac{3}{4}$ in. at widest part, and in some places it is three or four lines deep. The scar extends a little across the sagittal suture. The pulsations of the brain are distinctly felt through the cicatrix. About two years ago (1867) a carriage passing over him, he was kicked in the back, and his spine "broken." After this accident he was able to walk, though with great difficulty. In January, 1870, felt "rheumatic pains all over," and in the spring went into Bellevue Hospital. The legs felt "dead" to him, and no jerking was present. While in Bellevue had more or less retention of urine, and the catheter was sometimes used. The right leg was flexed for five weeks, the left never, but both were stiff and strongly adducted.

Condition on July 7th, 1871. Lies in bed; can raise each heel four or five inches

* It may be interesting to state that the patient's brother, an apparently healthy man, has similarly small pupils.

from bed, and can voluntarily move every articulation of lower limbs. Strength seems to be perfect in lower extremities; voluntary adduction being almost impossible to overcome. Reflex movements are very violent. In the erect position the movements of the legs are but little subordinate to the will. The patient can hardly stand even with the support of crutches; when movement is attempted but slight motion is seen at knee-joint. [I may here add, that I remember most vividly what is not sufficiently entered in the record, viz., the patient's very peculiar attempt at walking. He would get out of the bed with help, his legs being moderately rigid, but the moment his bare feet touched the floor most severe reflex movements occurred, producing tetanic rigidity of the limbs. Holding on by the head of the bed, a chair, or an attendant's arm, he could take a few steps, which consisted in sliding of the feet a little way, no hip or knee or ankle movement being apparent. He was also conscious of a tendency to adduction and crossing of legs.] Sensibility seems in all respect to be good (increased?) in lower limbs: and patient feels some numbness, more on right side. Upper extremities in normal state; bears marks of bed-sores on hips and sacrum. Spine exhibits cyphosis in its middle dorsal region, accompanied by slight scoliosis to the left. The greatest angular curvature is on level of fifth and sixth dorsal vertebrae. No difficulty in making water; bowels costive. At a later period the patient died of extensive erysipelas, but no autopsy could be obtained of the friends. I had diagnosed compression of the spinal cord by the products of broken-down vertebrae; there having been a traumatic Pott's disease. At one time there must have been much localized meningitis and perhaps superficial myelitis.

CASE III.—J. A., male, aged 38 years, admitted to the Epileptic and Paralytic Hospital, September 8, 1872. At first a painter, but during the last few years following the sea. Never had constitutional syphilis, rheumatism, or painter's colic. In October, 1870, while on a voyage from Italy to New York, he had an attack, which he thought was rheumatic, caused by constant exposure and overwork. His symptoms were severe pain in lower extremities, and a sensation as if a heavy weight were attached to all parts below the waist and were dragging him down. Has since had much pain, principally in left thigh, and has gradually lost use of lower limbs. In March, 1872, he was attacked at night with trembling of left upper extremity, accompanied by a feeling of numbness, and he has since progressively lost the power of moving his left hand. About September 1st, similar symptoms (trembling and numbness and subsequent palsy of hand) affected the right arm. At the same time a pain appeared in the left fifth intercostal space, a little outside the nipple, and this pain has remained. No head symptoms excepting occasional attacks of dizziness. Shortly before admission patient began to experience trouble in retaining his urine. It did not dribble away, but after a small quantity had accumulated in the bladder the desire to urinate was irresistible and the viscus was often suddenly and involuntarily emptied. At the beginning of November, 1872, I made an examination of this patient. The mouth is notably drawn downward on the right side; tongue projects straight out; the orbicularis oris cannot be fully contracted, so that whistling is very imperfectly done. No stammering, but speech is a little thick. Pupils normal, and sensibility good on all parts of head and face. The upper extremities exhibit paresis at shoulders and elbows, with very marked atrophy of many muscles of the hand, those of the thenar and hypothenar eminences, and nearly all the interossei. Fibrillary movements are distinct in many muscles of the upper trunk and arms. Sensibility in its various modes is perfect. There is no atrophy, paresis, or anæsthesia about trunk.

The lower extremities present peculiar symptoms. The patient cannot stand erect with his bare feet upon the floor; with his shoes on he can stand with help of nurse, and by great effort. During the attempt he oscillates greatly. While walking with help, slides both feet along in an equally awkward way; does not jerk or drag them. During these efforts, standing or walking, the muscles are rigid. Patient says that his legs feel stiff. Closing eyes does not make the attempt at standing worse. Examined in the recumbent posture, all movements are performed by lower limbs with effort, but with perfect co-ordination. Efforts cause more or less clonic spasms in lower limbs, and a pain in masses of muscle forming anterior part of thighs. Scratching soles, or other modes of irritation (cold impressions especially) produces strong reflex movements, mostly clonic. Strength at various joints perfect. Fibrillary movements in muscles are excited by tilting the skin. The various modes of sensibility are normal. The patient has observed that his legs stiffen when the cold air first strikes them on getting out of bed. The bladder trouble is as above described, an incontinence through spasm. The diagnosis of tumor compressing the spinal cord (incipient Pott's disease?) is made from co-existence of referred pain to legs and left side, of false paraplegia (tetanoid), and atrophy of a few muscles of upper extremity. This last symptom is explicable by compression of some anterior roots of nerves. The seat of compression is probably at the upper part of the cervical enlargement.

CASE IV.—J. R., aged 42 years, a baker, of intemperate habits, and admitting great sexual excesses, in 1864 had primary sore and secondary syphilitic symptoms. During the fall of 1869 he suffered from pain in the right side, and later, in the back; this being made worse by motion. March 6th, 1870, he awoke with both legs numb, accompanied by retention of urine. Admitted into the Epileptic and Paralytic Hospital, bearing a large bed-sore on sacrum, and having a paraplegia characterized by numbness and excessive reflex action. Before admission reports that he could not move lower limbs in the least. Improvement began in July; noticed sensation of distended bladder, acquired some voluntary movement of both legs; more control over left. Was unable to control rectum and bladder. Improved much under hypodermic injections of strychnia; bed-sore healing. During 1871-2 had iodide of potassium in large doses. I was inclined to consider the paraplegia one dependent upon a syphilitic lesion of the spinal dura mater, causing pressure and irritation. Discharged in early summer of 1872, able to walk with crutches, and gaining. Examination in supine position shows that voluntary movements of left lower extremity are very free; can raise foot more than twenty inches from the bed. Right foot can be raised only about ten inches. Strength at various joints (resistance to passive movements) normal. Has much twitching and spasm in lower limbs. Appreciates surface contact and tickling, but does not localize in pressures correctly. Senses of temperature and of pain normal. Reflex movements produced by examination of sensibility. Both legs feel numb below knees. Patient bears large node on the right tibia.

Re-admitted in fall of 1872. Examination January 5th, 1873. Walks with help of crutches, or of a stick. Steps small, legs tend to cross one another (adduction), and the lower extremities are apparently stiff. Patient has noticed great spasm in them at times, on standing up. In supine position voluntary movements of the left lower limb are normal in extent; flexion of thigh and knee on right side is limited by stiffness at knee and hip joints. Power of resistance at both knee-joints normal; as also at other joints of lower limbs. Reflex excitability increased, more on the left than on right side. Co-ordination of movements (eyes

closed) quite perfect. Is conscious of passive movements at knee-joints; unconscious of them when made at ankles and toes. Sensibility preserved except as regards the tactile sense, which is much impaired on feet.

The patient states that his bad walking is due to stiffness of the limbs. When he attempts to stand alone in bare feet, the reflex spasm is so great as to cause him to lose his balance; with help, and in stockings and shoes, can walk as above described. Has better control over his legs some days than others. His urine does not dribble away nor is it retained, but when the desire to urinate is felt he must empty the bladder almost immediately, or the urine is forced out against his will. Is improving while taking large doses of iodide of potassium.

CASE V.—J. K., male, aged 36, admitted to the Roosevelt Hospital, service of Dr. W. H. Draper, February 9, 1872. In March, 1871, patient began to suffer from pain in his spine, about the level of the third and fourth dorsal vertebrae; and about the middle of April he noticed a tumor, about as large as a small hen's egg, in the same locality. The tumor grew and the pain became more severe until May, when the application of a plaster is said to have caused both pain and swelling to disappear.

Late in December, the pain and tumor coincidently returned, and about one month ago, patient observed weakness in his legs, most marked in the right. The tumor has been stationary since the beginning of the year. Examination of upper limbs reveals nothing abnormal. Over third dorsal vertebra is a firm, slightly red-denied tumor, about the size of half an egg; this tumor is painful when firmly pressed. Intercostal muscles act very slightly; respiration mostly abdominal; marked impairment of sensation, motion, and co-ordination in the lower limbs; worse on right side; bowels torpid. When he wishes to urinate, he is obliged to do so at once. February 17, is much troubled by reflex contractions. Examined by Dr. Seguin, at Dr. Draper's request. Marked loss of sensibility in right leg; lessened sensibility in left. Co-ordination in both legs impaired. Resistance to flexion of knee nearly normal while lying on back, but when upright this is notably lessened. I have a very clear remembrance of this examination, and will add to the short entry made at the hospital. The "in-co-ordination" consisted in extraordinary stiffness of the lower limbs, when the patient attempted to walk with the aid of a nurse. At the same time the feet tended to cross each other from strong action of adductor muscles. In the recumbent posture no trace of in-co-ordination appeared, and there was almost perfect strength in all parts of the lower extremities. It was this wonderful contrast between seeming extreme paraplegia when standing or attempting to walk, and the preservation of motor power when lying on back, that caused surprise to all present. At the same time I made out a decided angular spinal curvature involving the third and fourth dorsal vertebrae, and decided that the case was one of Pott's disease of the spine. February 20th, more pain in tumor. Incision causes escape of blood only. March 1st, retention of urine has appeared; catheter used. Angular curvature more pronounced. March 10th, reflex movements in legs very annoying; great dyspnoea. April 10th, urine dribbles away; bed-sores forming over sacrum and trochanters. Spinal angle increasing. During succeeding months the bed-sores extended greatly, the spinal curve became more acute, and power in legs was reduced to a minimum. After living for a month and a half at the point of death, patient expired July 9th.

I am indebted for the above history to Dr. N. B. Sizer, House Physician to the Presbyterian Hospital; and wish to express my thanks to Dr. Robert F. Weir, Surgeon to the Roosevelt Hospital, for permission to make use of the case.

ARCHIVES

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Amaurosis and Deafness in Diseases of the Brain.

Rational Treatment of Poisoning by Organic Substances.

On the Various Kinds of Hemiplegia.

On Counter-Irritation: Its Importance and Rational Use

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